

# Intermittent AV conduction disturbances in patients with AV nodal bypass tracts<sup>1</sup>

## *Possible mechanisms of unusual variant of tachycardia-bradycardia syndrome*

AGUSTIN CASTELLANOS, RUEY J. SUNG, BENJAMIN BEFELER, ALVARO MAYORGA-CORTES, CESAR CONDE, STEPHEN M. MALLON, AND ROBERT J. MYERBURG

*From the Division of Cardiology, Department of Medicine, University of Miami School of Medicine, and the Veterans Administration Hospital, Miami, Florida, U.S.A.*

*His bundle recordings were performed in 2 patients in whom AV nodal bypass tracts coexisted with intermittent AV conduction disturbances occurring below the site from which the His bundle deflection was recorded. Case 1 had: (a) tachycardia dependent right bundle-branch block, (b) persistent HV prolongation, and (c) bradycardia dependent AV block. Case 2 showed: (a) intra-atrial conduction delay, (b) tachycardia dependent left bundle-branch block with HV prolongation, (c) bradycardia dependent HV conduction disturbance, (d) tachycardia-bradycardia syndrome of an unusual type; the latter presumably resulted, during atrial flutter, from the alternation of rapid AH conduction through the bypass tract with intermittent (complete) distal His bundle block or bilateral bundle-branch block.*

In a previous communication in this journal (Befeler *et al.*, 1976), we discussed the occurrence of bundle-branch block and normal PR intervals in patients with AV nodal bypass tracts. It appeared justified, in view of the very few existing publications (Krishnaswami and Geraci, 1974; Brechenmacher, 1975), to report the association of AV conduction disturbances with AV nodal bypass tracts (in absence of coexisting Mahaim tracts). In addition the possible mechanisms of an unusual type of tachycardia-bradycardia syndrome are discussed.

### Subjects and methods

His bundle recordings were obtained in 2 patients referred to the Cardiovascular Laboratory for evaluation of recurrent supraventricular tachycardias. The procedure was explained and informed consent obtained. The normal values in our department for the various conduction intervals are as follows (Castellanos *et al.*, 1975; Befeler *et al.*, 1976): PA=20-45 ms; AH=55-120 ms; HV=35-55 ms.

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### Results

#### CASE 1

This 52-year-old man with an old anterolateral wall myocardial infarct has already been described elsewhere (Befeler *et al.*, 1976). During sinus rhythm (Fig. 1, first beat) the PR interval was normal (165 ms). The HV interval was prolonged (60 ms) and the QRS complexes showed a right bundle-branch block (RBBB) pattern.

Although the AH interval was only at the lower limits of normal (60 ms) the presence of an electrophysiological AV nodal bypass was suggested by: (a) persistence of 1:1 AV conduction at rates up to more than 200/min, with minimal (35 ms) increase in the AH interval; and (b) a dual pathway response during atrial pacing with the extrastimulus technique (Denes, Wu, and Rosen, 1974a).

Towards the end of the procedure bradycardia dependent AV block was exposed by the post-extrasystolic pause following an electrically induced premature atrial impulse (second beat (Fig. 1)) (Coulmel *et al.*, 1971; Castellanos *et al.*, 1975). Whereas, AV conduction occurred with HH intervals between 570 and 695 ms, block below the H deflection was seen when this interval increased to

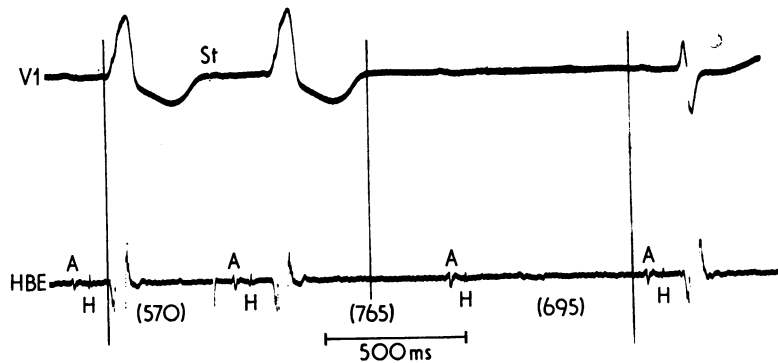


Fig. 1 Case 1. AV nodal bypass tract coexisting with: (a) persistent HV prolongation in conducted beats, (b) tachycardia dependent right bundle-branch block, and (c) bradycardia dependent HV block. The first, third, and fourth P waves are of sinus origin. St=premature stimulus delivered to the atria. A=atrial deflection recorded by the His bundle electrographic lead (HBE). H=His bundle deflection. All intervals are expressed in ms.

765 ms. In addition, disappearance of the bundle-branch block pattern in the last conducted beat suggested that tachycardia dependent right bundle-branch block was also present (Rosenbaum *et al.*, 1973).

In Fig. 1 all conducted beats (regardless of QRS duration) had similar AH and HV intervals. These observations indicate that there was a conduction delay in the distal His bundle below the site from which the H deflection was recorded (Befeler *et al.*, 1976). The bradycardia dependent conduction block could have been either in the same site (distal His bundle) or within the left bundle-branch.

To summarise, in this case, an electrophysiological AV nodal bypass tract coexisted with persistent HV prolongation and bradycardia dependent

AV block. Thus, pacemaker implantation was recommended.

#### CASE 2

A 63-year-old man with a documented history of recurrent atrial flutter and atrial fibrillation had a short PR interval with narrow QRS complexes (Fig. 2, top strip). Occasional atrial premature beats were conducted with a normal PR interval and a left bundle-branch block pattern.

This patient was brought to Jackson Memorial Hospital by the Fire Rescue Squad because of palpitation and dizziness of three hours' duration. The tracing obtained on arrival (Fig. 2, second strip) showed a tachyarrhythmia with wide and irregular QRS complexes occurring at a rate ranging be-

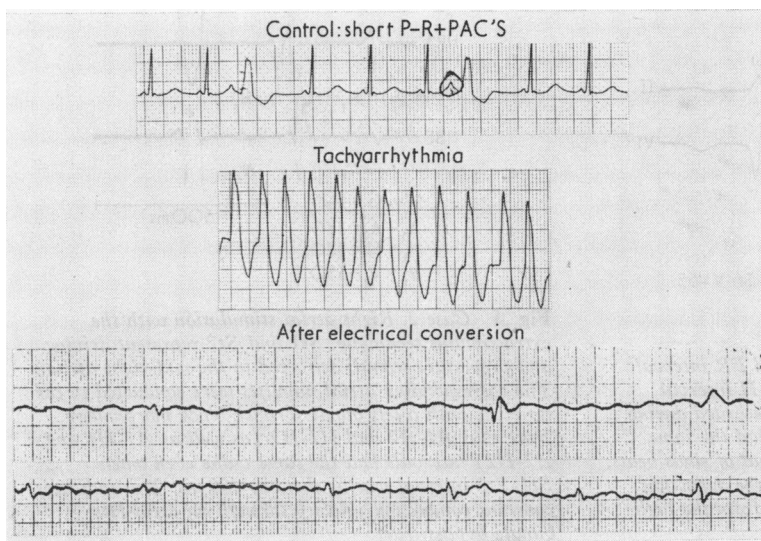


Fig. 2 Case 2. AV nodal bypass tract associated with: short PR interval and spontaneous premature atrial beats with tachycardia dependent left bundle-branch block (top strip), 'pseudo-ventricular tachycardia' caused by atrial flutter or fibrillation coexisting with tachycardia dependent left bundle-branch block (second strip), and atrial flutter coexisting with complete AV block (third and fourth strips) induced or exposed by cardioversion.

tween 205 and 235/min. Because the arrhythmia was interpreted as ventricular tachycardia the patient received two transthoracic synchronised electrical discharges of 200 and 300 Ws, respectively. Since these were ineffective an additional 400 Ws discharge was given. The rhythm strip obtained immediately afterwards showed atrial fibrillation (or flutter) with complete AV block (Fig. 2, third strip). This was treated with an infusion of isoprenaline (Fig. 2, bottom strip) followed by emergency pacemaker insertion.

Twenty-one hours after admission the electrocardiogram showed sinus tachycardia with normal PR interval and left bundle-branch block, which disappeared when the rate decreased to below 100/min. The patient did not have clinical, electrocardiographic, or enzymatic evidence of acute transmural myocardial infarction. The x-ray films showed a normal heart size with calcification in the aortic valve area.

His bundle recordings obtained during sinus rhythm at a rate of 83/min (Fig. 3, left) showed an intra-atrial conduction disturbance manifested by wide (125 ms) P waves and prolonged PA intervals (55 ms). Whereas the PR interval and HV interval were normal (150 and 55 ms, respectively), the AH interval was short (40 ms). The latter suggested the presence of an electrophysiological AV nodal bypass tract (Castellanos *et al.*, 1971; Coumel *et al.*,

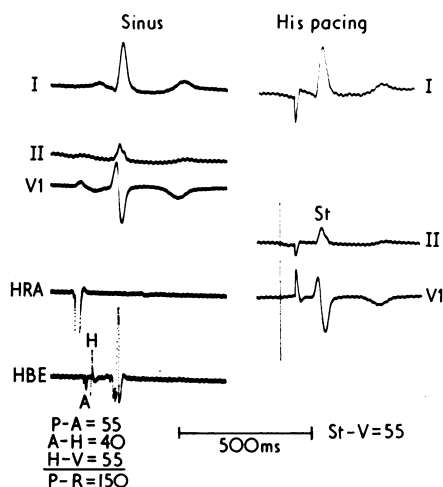


Fig. 3 Case 2. Sinus beat with normal PR interval and narrow QRS complex with short AH interval (left-sided panel). The St-V interval recorded during His bundle pacing (right-sided panel) had the same value (55 ms) as that of the HV interval of sinus beats. This validates the His bundle origin of the deflection labelled H. HRA=bipolar electrogram from the high right atrium.

1972; Caracta *et al.*, 1973). The His bundle origin of the corresponding deflection was validated by His bundle pacing (Fig. 3, right) (Narula, Scherlag, and Samet, 1970).

Mid-right atrial stimulation showed development of left bundle-branch block at a rate of 100/min. 1:1 conduction occurred up to a rate of 150/min when the atria failed to respond. Thus, occurrence of second degree AV block could not be demonstrated.

Pacing with the extrastimulus technique (Wit *et al.*, 1970) was performed at a basic cycle length of 585 ms. Driven QRS complexes (first beat in Fig. 4) had a left bundle-branch block pattern with prolonged St-A and HV intervals (70 and 80 ms, respectively). These values corroborated the existence of an intra-atrial conduction disturbance and suggested an additional conduction delay in the distal His bundle or right bundle-branch, which was revealed by pacing. The A1-A2 and St1-St2 intervals were identical while the latter were decreased from 560 to 320 ms; thereafter the A1-A2 intervals maintained a value of 320 ms until the effective refractory period of the atria was reached (240 ms).

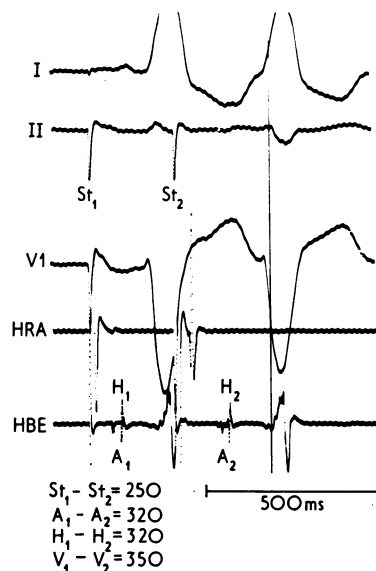


Fig. 4 Case 2. Right atrial stimulation with the extrastimulus technique. St1 and St2 represent driving and premature stimuli delivered to the mid-right atrium. The corresponding atrial impulses were conducted to the ventricle with a left bundle-branch block morphology. Note that A1-A2 and H1-H2 (as well as A1-H1 and A2-H2) intervals had the same value even when St1-St2 interval was as short as 250 ms. This indicates constant conduction velocity through an atrio-His bundle bypass tract.

Plotting the A1–A2 intervals against the H1–H2 and V1–V2 intervals indicated that the first two values were identical throughout, but that V1–V2 intervals exceeded H1–H2 intervals at A1–A2 intervals shorter than 350 ms (Fig. 4). The results of pacing with the extrastimulus technique suggested that as the St1–St2 intervals were decreased, St2 showed (by comparison with St1) progressive increases in intra-atrial and His–Purkinje (distal His bundle and/or right bundle-branch) conduction times, with constant conduction velocity through the AV nodal bypass tract.

Two different types of rate-dependent conduction disturbances are shown in Fig. 5. The HV intervals were unchanged from control value when the HH intervals measured 695 ms. A tachycardia dependent left bundle-branch block (associated with an HV conduction delay, also tachycardia dependent) appeared when HH was reduced to 350 ms. In contrast, a bradycardia dependent conduction disturbance occurred when HH increased to 795 (because of the post-extrasystolic pause). The fourth supraventricular impulse, after having reached the His bundle, was either: (a) completely blocked within the His bundle and followed by a distal His bundle, or proximal right bundle, escape beat (fifth H deflection); or (b) conducted with a significant delay within the His bundle (resulting in 'split' H deflections).

To summarise, this symptomatic patient with recurrent supraventricular tachyarrhythmias had an AV nodal bypass tract associated with tachycardia and bradycardia-dependent His–Purkinje conduction disturbances. Therefore, pacemaker implantation was recommended.

## Discussion

### ATRIO-HIS BUNDLE BYPASS TRACT WITH NORMAL PR INTERVAL

The findings illustrated in Fig. 1 to 5 confirm that the essential electrophysiological feature of an AV

nodal bypass tract in patients with recurrent supraventricular tachyarrhythmias is the presence of a short (or at the lower limits of normal) AH interval which shows an abnormal response to atrial stimulation at increasing rates and/or with the extrastimulus method (Coulmel *et al.*, 1972; Castellanos *et al.*, 1973; Caracta *et al.*, 1973; Bissett *et al.*, 1973; Denes *et al.*, 1974b; Befeler *et al.*, 1976).

Although classically the PR intervals are short in these cases, a normal PR interval can occur (Fig. 1 and 3), if there are coexisting His–Purkinje and/or intra-atrial conduction delays (Brechenmacher, 1975; Narula, 1975; Befeler *et al.*, 1976).

The term AV nodal bypass tract as used in this communication implies the presence of a rapidly conducting electrophysiological pathway because of which the atrial impulses reach the His bundle without having experienced the 'normal' quantitative or qualitative AV nodal delay.

In Cases 1 and 2 no inferences could be drawn as to whether these bypass tracts were (anatomically) intranodal (Moe, Preston, and Burlington, 1956; Goldreyer and Bigger, 1971; Rosen, Mehta, and Miller, 1974), atrio–His bundle (of the type described by Brechenmacher *et al.* (1974) and Brechenmacher (1975)), or atrio–AV nodal (so-called) James' fibres or transitional fibre (James, 1961; Anderson *et al.*, 1975).

It should be stressed that in Fig. 1, 3, and 5 the interval elapsing between the inscription of the atrial deflection (recorded from the vicinity of the AV node) and the H deflection did not represent 'normal' AV nodal conduction time (Castellanos *et al.*, 1971). Rather it reflected differences in arrival of excitation at these sites (Aranda *et al.*, 1976). For example, part of the increase in the 'AH interval' which occurs in patients with AV nodal bypass tracts at decreasing cycle lengths and pacing from different sites can be ascribed to varying degrees of intra-atrial conduction delay and varying modes of entry into both bypass tract and AV node (Aranda *et al.*, 1976). These factors play a similar role in the

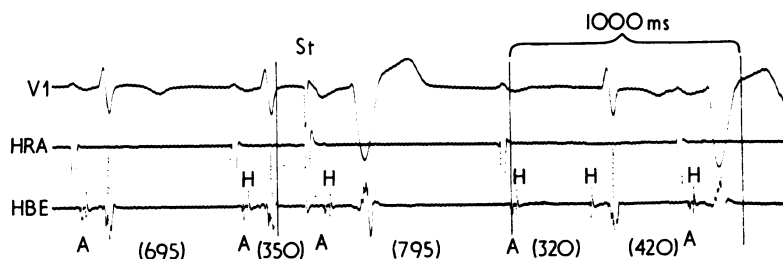


Fig. 5 Case 2. AV nodal bypass tract associated with: (a) coexisting tachycardia dependent left bundle-branch block and HV prolongation, and (b) bradycardia dependent conduction disturbance occurring below the site from which the H deflection was recorded.

AV conduction patterns occurring in patients with Kent tracts (WPW syndrome) (Denes *et al.*, 1974b; Gallagher *et al.*, 1975; Wellens, 1975; Castellanos *et al.*, 1976). Thus, the constant AH intervals observed in Case 2 can be explained by assuming that all supraventricular impulses entered an atrio-His bundle tract of the type described by Brechenmacher at a site close to which the A deflection was recorded.

#### INTRAVENTRICULAR BLOCK COEXISTING WITH AV NODAL BYPASS TRACT

Patients with AV nodal bypass tracts (not associated with Mahaim tracts) usually have narrow QRS complexes because, as first suggested by Burch and Kimball in 1946, the supraventricular impulse reaches the ventricular muscle through a normal His-Purkinje system. However, wide QRS complexes occur when the clinical course is complicated by coronary artery disease (as in Case 1) or primary conducting system disease (as in Case 2). Unilateral bundle-branch involvement results in isolated right or left bundle-branch block (Bissett *et al.*, 1973; Brechenmacher, 1975; Narula, 1975; Befeler *et al.*, 1976). Additional intra-His bundle and/or contralateral bundle-branch disease (Fig. 1 and 5) can produce symptomatic, persistent or paroxysmal (rate dependent), AV conduction disturbances requiring pacemaker implantation (Fig. 1 to 5). The latter combination explains the (apparent) paradox of 'accelerated A-V (or more properly A-H) conduction associated with complete A-V block' (Krishnaswami and Geraci, 1974).

#### UNUSUAL VARIANT OF TACHYCARDIA-BRADYCARDIA SYNDROME

Case 2 had, on arrival at the hospital, an atrial tachyarrhythmia (flutter or fibrillation) with wide irregular QRS complexes resulting from the coexistence of an AV nodal bypass tract with tachycardia-dependent left bundle-branch block (Fig. 2). Conduction time through the His bundle and right bundle-branch was probably normal since otherwise the ventricular rate would not have exceeded 200/min. Though His bundle electrograms were not recorded at that precise moment, deductive reasoning based on surface electrocardiographic and subsequent intracardiac electrophysiological studies (Fig. 2 to 5) supports these assumptions.

The term 'pseudoventricular tachycardia' (Herrmann *et al.*, 1957) was originally applied to an arrhythmia occurring during atrial flutter and fibrillation in some patients with Wolff-Parkinson-White syndrome (presumably those with a Kent tract having a short effective refractory period (Castellanos *et al.*, 1973)). However, Fig. 2 shows

that, as previously reported by Brechenmacher *et al.* (1974), a similar electrocardiographic pattern can be observed in the presence of an AV nodal bypass tract.

The sudden appearance of AV block following three high energy transthoracic electrical discharges was probably the result of an acute injury affecting the distal His bundle and/or right bundle-branch, related to or exposed by cardioversion. This sequence of events resulted in an unusual variant of tachycardia-bradycardia syndrome since the ventricular rate changed abruptly from very rapid to very slow.

Pacemaker implantation was recommended in both patients, because they were clinically symptomatic and had rate related bundle-branch block associated with either persistent or tachycardia dependent HV prolongation and bradycardia dependent AV block.

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Requests for reprints to Professor Agustin Castellanos, Division of Cardiology, University of Miami School of Medicine, P.O. Box 520875, Miami, Florida 33152, U.S.A.